Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION

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Circulation 1994;90;2581-2590
Circulation is published by the American Heart Association. 7272 Greenville Avenue, Dallas, TX 72514
 Print ISSN: 0009-7322. Online ISSN: 1524-4539

AHA Medical/Scientific Statement

Special Report

Active and Passive Tobacco Exposure: A Serious Pediatric Health Problem

A Statement From the Committee on Atherosclerosis and Hypertension in Children, Council on Cardiovascular Disease in the Young, American Heart Association

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Cigarette smoking and passive exposure to tobacco smoke are important causes of mortality in the United States. Active and passive exposure to tobacco smoke are projected to contribute to more than 400 000 deaths annually.[1,2] Coronary artery disease, cancer (particularly lung cancer), and chronic obstructive lung disease are the major sequelae of smoking in adults. Because nearly all smokers begin smoking before the age of 18 years, and large numbers of children are beginning to smoke at the age of 10 or 11, prevention is a major public health goal in the United States.[3] Currently, about 28% of high school seniors have smoked in the last month, a decline from the peak smoking rates of the 1970s but essentially unchanged over the last 5 years. The highest rates are seen in those with the lowest socioeconomic status.[4]

Primary prevention of smoking is essential because nicotine is one of the most highly addictive substances available.[5,6] Nicotine meets all the criteria that define an addictive substance: it produces brief, pleasurable psychoactive effects; its use occurs despite the known harmful effects; tolerance to both the pleasurable and unpleasant effects develops during early usage; higher

"Active and Passive Tobacco Exposure: A Serious Pediatric Health Problem" was approved by the Science Advisory Committee of the American Heart Association on June 16, 1994.

Requests for reprints should be sent to the Office of Scientific Affairs, American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231-4596.

doses overcome tolerance; and withdrawal symptoms occur when the substance is no longer used. At least 40% of people who have ever smoked a cigarette believe they are physically dependent on tobacco products. By contrast, only 6% to 8% of those who have ever drunk alcohol have recently binged or have felt dependent, and only 4% of those who have ever used cocaine feel dependent. Risk factors for initiating smoking include use by other family members and friends, peer approval, low socioeconomic status, poor academic achievement, poor self-image, and susceptibility to influence of others and advertising images that project smoking as pervasive and glamorous.[7]

The Surgeon General's report on cigarette smoking and children has emphasized the epidemiology and substantial morbidities of tobacco use by children and adolescents.[4] Hazards to children include

- Increased neonatal and infant mortality in children whose parents smoke
- Increased morbidity from respiratory disease in children exposed to tobacco smoke
- Adverse physiological and metabolic changes in adolescents who smoke[4,5]

Most important, atherosclerosis, endothelial and epithelial injury, and altered lung function—the initiating pathophysiological events that lead to coronary artery disease, cancer, and chronic obstructive pulmonary disease—have been described in youths who use or are exposed to tobacco products.[8-10]

This scientific statement summarizes the current literature on the harmful effects of exposure to tobacco among youth. The following areas are addressed specifically:

- Cardiovascular morbidity
- Respiratory morbidity
- Low birth weight
- Infant mortality
- Effects of smokeless tobacco
- Interaction of smoking with other adverse lifestyles and behaviors

Finally, current interventions to prevent smoking by children and their parents are discussed.

Cardiovascular Morbidity

Acute cardiovascular effects of smoking include tachycardia, increased blood pressure, decreased exercise tolerance, coronary vasoconstriction, elevated blood carboxyhemoglobin concentration, and increased tendency to thrombosis.[11-13] This section focuses on the chronic and sustained physiological, metabolic, and pathological sequelae of cigarette smoking.

An immediate effect of cigarette smoking is an increase in the carboxyhemoglobin concentration.[14] Carbon monoxide produced by smoking binds tightly to hemoglobin and displaces oxygen from available binding sites,[15] which leads to a net reduction in systemic oxygen transport. Adults who smoke have higher hemoglobin concentrations, which are believed to be a compensatory mechanism for this phenomenon.[12,14] Increased 2,3-diphosphoglycerate concentration, a substance that alters oxygen's affinity for hemoglobin,[15] can be correlated with the concentration of thiocyanate, a measure of smoke, in children's blood.[16] Thus, both active and passive exposure to tobacco smoke have a deleterious effect on oxygen transport in children.

The adverse physiological effects of smoking have been demonstrated in two evaluations from the CARDIA study of young adults. Chronic smokers (5 to 7 years' average duration) who had not smoked for 2 to 8 hours before exercise testing had blunted heart rate responses to exercise and diminished exercise tolerance compared with nonsmokers. Maximal heart rate in smokers was decreased by 4% and exercise test duration by 7% compared with nonsmokers.[12] It was speculated that these effects are secondary to downregulation of cardiac beta receptors following to exposure to tobacco smoke. In clinical and echocardiographic studies conducted 5 years later, smokers had higher heart rates, increased left ventricular mass, and elevated right ventricular and left ventricular afterload.[17]

Consistent changes in serum lipoproteins have been demonstrated both in children passively exposed to tobacco smoke and in young adult smokers. Children who smoke, when compared with those who do not smoke, have higher levels of triglycerides, very-low-density–lipoprotein (VLDL) cholesterol, and low-density lipoprotein (LDL) cholesterol, and lower levels of high-density lipoprotein (HDL) cholesterol.[18] These differences are comparable to those seen in adults who smoke. A decline of 3 to 5 mg/dL in HDL cholesterol has been found in children passively exposed to tobacco smoke compared with children not exposed to tobacco smoke.[16] These lipid levels put children who smoke and those who are exposed to smoke in jeopardy, because they are at risk for smoking and adverse lipoprotein profiles.

Smokers have increased platelet aggregation compared with nonsmokers.[11,19] Increased platelet aggregation also occurs when a nonsmoker smokes or is passively exposed to smoke. Thus, this increased thrombotic potential is found in children exposed to passive smoke and in adolescents who are beginning to smoke.

Key evidence linking cigarette smoking to atherosclerosis has come from the Pathobiological Determinants of Atherosclerosis in Youth (PDAY) study.[8] Investigators correlated biochemical markers of tobacco smoke exposure (blood thiocyanate and cotinine levels) to coronary artery and arterial lesions in young adults aged 15 to 35 years who died accidentally. Cigarette smoking increased the risk of having raised atherosclerotic plaques in all vascular beds studied, including the right coronary artery and descending aorta. The likelihoods were greatest for the descending aorta, which is of interest because abdominal aortic aneurysms in adults are strongly associated with smoking. The likelihood of having raised coronary arterial lesions in smokers was greater than the likelihood attributed to a 50 mg/dL increase in VLDL and LDL cholesterol and a 20 mg/dL decrease in HDL cholesterol.

Cigarette smoking causes endothelial injury, thought to be a primary initiating event of atherosclerosis.[20-23] This has been demonstrated in several ways, including ultrastructural changes in aortic endothelial cells, endothelial cell turnover, and endothelial cell function.[21-23] These pathological changes have been observed in the umbilical arteries of infants born to mothers who smoke[22] and after passive exposure to smoke.[24]

Respiratory Morbidity

Tobacco smoke and its products affect the lungs and respiratory tracts of infants, children, and adolescents by passive exposure in utero caused by maternal smoking, by passive exposure to tobacco smoke produced by parents and caretakers, or by active exposure caused by smoking tobacco products. Active cigarette smoking is the major cause of chronic obstructive lung disease and chronic respiratory symptoms in the United States.[25] Active smoking by healthy adults also leads to reductions in lung function.[26] Similarly, increases in respiratory symptoms[27] and reductions in lung function[28] have been described in children who smoke. Children who smoke and have consisent respiratory illness, such as asthma, have the greatest decreases in lung function.[28]

Environmental tobacco smoke, a complex mixture of exhaled mainstream smoke and noninhaled, sidestream smoke, also contributes to respiratory morbidity of children.[29] Tobacco combustion produces multiple toxic compounds.[30] Although environmental tobacco smoke differs from mainstream smoke in several ways, it contains many of the same toxic substances. Infants and toddlers may be especially at risk when exposed to environmental tobacco smoke.[31] Exposure to toxic compounds in infancy is particularly problematic because early lung development appears to be a critical determinant of respiratory health.[32]

Respiratory infections are frequent in childhood, and about 30% of all infants are treated by a physician for bronchiolitis, croup, or pneumonia.[33] Risk of respiratory illness is increased in infants and children whose parents smoke.[34-37] Infants exposed to maternal smoking had an increased incidence of lower respiratory tract infection.[38] This effect showed a dose-response relationship to maternal smoking and decreased after the first year of life. Infants with bronchiolitis before the age of 2 years were 2.4 times more likely to have been exposed to maternal smoking than infants who did not develop a lower respiratory tract infection.[39] Wright and coworkers[40] found that infants whose mothers smoked at least one pack per day had 2.8 times the risk of developing a lower respiratory infection. Children hospitalized for acute lower respiratory illness before age 2 are 1.8 times as likely to live with smokers than control subjects hospitalized for nonrespiratory illness.[41] Considering the substantial morbidity, and even mortality, of acute respiratory illness in childhood, a doubling in risk attributable to passive smoking clearly represents a serious pediatric health problem.[31]

The manner in which passive exposure to environmental tobacco smoke leads to increased lower respiratory infection risk is unknown. Prenatal effects of maternal smoking on the lungs have been demonstrated by Hanrahan and coworkers,[42] who found that infants born to mothers who smoke have reduced forced expiratory flows. The degree of reduction was correlated with increasing maternal urine cotinine/creatinine ratios during pregnancy. Subsequent lung dysfunction and respiratory illness[32] could thus begin by in utero exposure to maternal smoking, with alteration of the developing lung. Postnatal exposure to environmental tobacco smoke may also increase the risk of lower respiratory tract illness. Exposure to postnatal, paternal smoking alone, without in utero exposure, is associated with increased hospitalization of children for respiratory illness.[43] Children whose mothers smoked only after pregnancy were still more likely to develop acute respiratory illness.[44]

The Surgeon General's report[10] on the health effects of passive smoking and the report of the National Research Council[31] both concluded that maternal smoking reduces lung function in young children. Kauffmann and coworkers[45] found a reduction of forced expiratory volume in 1 second (FEV_1) for 10 mL/g of tobacco per day smoked by the mother. Several other studies have also shown reductions in lung function.[46-50]

The effect of environmental tobacco smoke exposure depends on the dose. In a population-based longitudinal study of lung function from 5.5 to 25 years in Tucson, researchers were unable to demonstrate an effect of passive smoke exposure.[51] By contrast, a study of subjects of similar age in East Boston demonstrated reduced forced expiratory flows associated with maternal smoking.[52] Collaborative analyses of the two data sets[53,54] by these groups confirmed the lack of impact in Tucson and reduced flow associated with maternal smoking seen in Boston. These geographic disparities could be due to higher exposure levels in Boston, where the homes may be built in such a way that there is less air exchange compared with Tucson.

Asthma is a leading chronic childhood illness in the United States. Morbidity and mortality due to asthma have increased in recent years, particularly in children.[55-57] Exposure to environmental tobacco smoke in childhood is associated with an increased risk for developing asthma among certain children at risk. Children aged 0 to 5 years who are exposed to maternal smoking are 2.1 times more likely to develop asthma compared with those free from exposure.[58] Risk of asthma is 2.5 times higher in children exposed to maternal smoking when the mother has less than 12 years of education.[59] In a questionnaire study of 3482 nonsmoking children, Burchfield and coworkers[60] found that asthma was increased in males when both parents had smoked compared with those whose parents were nonsmokers. In other studies researchers failed to find increased asthma risk with maternal smoking, but these studies were not controlled for dose or socioeconomic status.[61] Childhood exposure to environmental tobacco smoke seems to be a risk factor for development of asthma when the dose is higher and other risk factors such as low socioeconomic status are present. Environmental tobacco smoke has been associated with the development of asthma through immune mechanisms. Studies in Italian schoolchildren exposed to environmental tobacco smoke[62,63] have found increases in bronchial reactivity, IgE levels, eosinophilia, and sensitization to aeroallergens. Considering these findings and the strong relationship of atopy and IgE to the development of asthma,[64] environmental tobacco smoke may not only alter the developing lung's structure and function but also augment the exposed child's level of atopy and risk for asthma.

Exposure to environmental tobacco smoke has been associated with increased asthma-related trips to the emergency room and related costs.[65,66] Compared with healthy children, children with a history of wheezing or

asthma have increased airway reactivity[46,67] that is not explained by the acute effects of environmental tobacco smoke exposure.[68] Increased bronchial reactivity in asthmatic individuals may be due to the effects of chronic exposure to environmental tobacco smoke. Chilmonczyk and coworkers[69] recently demonstrated decreased lung function and increased exacerbation frequency in asthmatic children exposed to environmental tobacco smoke. The recent EPA report[29] states that "there is now sufficient evidence to conclude that passive smoking is associated with additional episodes and increased severity of asthma in children who already have the disease."

Low Birth Weight

Over the past several decades cigarette smoking during pregnancy has been associated with adverse pregnancy outcomes, including increased incidences of low birth weight.[5]

When the effects of maternal smoking are considered, a distinction must be made between low birth weight (ie, small for gestational age, defined as birth weight less than 2500 g in an otherwise normally mature term infant) and prematurity. This distinction is important because prematurity is strongly associated with increased risk of perinatal morbidity and mortality in the absence of maternal smoking. In several studies (after controlling for other factors), birth weight generally has been decreased by an average of approximately 200 g in infants whose mothers smoked throughout pregnancy.[70,71] This is associated with a twofold to fourfold greater relative risk of infants small for their gestational age born to mothers who smoked.

The mean duration of gestation is not affected by maternal smoking.[70,71] Therefore, premature delivery (ie, delivery before 37 weeks of gestation) is not associated with smoking. However, smoking is associated with a decrease in mean birth weight and thus an increase in proportion of lower birth weight infants at all gestational ages and infant mortality.

In most studies cessation of smoking early in pregnancy prevented the effects of low birth weight associated with smoking. In mothers who stopped smoking late in pregnancy (7 to 8 months), infants' mean birth weights were lower than those born to nonsmokers but higher than those of infants whose mothers smoked throughout pregnancy.[72] Mothers who smoked only during the first trimester had a 30% increased risk of having a low–birth-weight infant. Those who smoked during the first and second trimesters had a 70% increased risk, and those who smoked throughout pregnancy had a 90% increased risk of having a low–birth-weight infant.[70,71]

The effects of maternal smoking during pregnancy appear to be dose-related.[73] Mothers who were light smokers (less than 10 cigarettes per day) delivered infants weighing on average 96 g less than infants of nonsmokers. Mothers who were moderate (10 to 19 cigarettes per day) or heavy (20+ cigarettes per day) smokers delivered infants whose average birth weights were 183 g or 200 g, respectively, less than nonsmokers' infants. Rates of small-for-gestational-age infants increased from 3.4% in nonsmokers to 6.7% in light smokers to 8.2% in heavy smokers.[74] Maternal age, parity, alcohol consumption, and use of caffeine all interacted with smoking and increased risk for infants born small for their gestational age.[75]

Cigarette smoking may affect fetal growth by several mechanisms.[75] Some compounds found in tobacco smoke, such as nicotine, carbon monoxide, and polycyclic aromatic hydrocarbons, are known to cross the placenta. Some of these compounds have been identified in newborns of smokers and those exposed to exhaled tobacco smoke.[76] Carbon monoxide has the affinity to bind with hemoglobin to form carboxyhemoglobin, which reduces the capacity of the blood to adequately transport oxygen to the fetus. Smoking (ie, nicotine) probably causes vasoconstriction of the umbilical arteries and impedes placental blood flow. Ultrastructural changes of the placenta are found in smokers[77] and include thickening of the basement membrane, collagen increase in the villous stroma, and fewer fetal capillaries with smaller lumina. These changes may interfere with placental blood flow. The combination of intrauterine hypoxia and impaired placental blood flow is believed to slow fetal growth.[77] Although most studies associate fetal growth retardation with maternal smoking, some studies also show effects of paternal smoking. Even allowing for maternal smoking habits, paternal smoking was associated with a decline in infant birth weight of 112 g.[78]

Infant Mortality

Higher mortality occurs in infants of mothers who smoke compared with those who do not smoke.[70,71] This is true both for neonatal morbidity (the first month of life) and thereafter (1 month to 1 year). This higher risk of mortality is independent of other factors associated with mortality, including birth weight. Perinatal mortality rates are 25% to 56% higher in infants of mothers who smoke compared with those who do not smoke for every birth weight category.[79]

When the association of maternal smoking with age and cause of infant death was explored, parental smoking was a more significant risk factor for postneonatal deaths than for neonatal deaths.[80] The ratio was especially high for respiratory disease and sudden infant death syndrome. Deaths from these two postneonatal causes of infant mortality do not seem to be attributable to birth weight differences between infants of smokers and nonsmokers. Passive exposure of infants to maternal smoking has been documented by urine cotinine, an indicator of cigarette smoke absorption.[81,82]

The relation of maternal smoking to sudden infant death syndrome appears dose-dependent, [83] which suggests that respiratory deaths and sudden infant death syndrome may be related to the infants' exposure to smoke after birth.[80,84] Intrauterine exposure may also be important. The risk of sudden infant death syndrome was greater in infants exposed to tobacco smoke in utero and postnatally (threefold increase) than those with only postnatal exposure (twofold increase), compared with infants not exposed to smoke.[85]

Smoking may result in chronic fetal hypoxia, impairing normal development of the central nervous system.[84,86] Nicotine causes necrosis of cells in the brain stems of fetal Sprague-Dawley rats.[86] Nicotine-induced cell death may result from direct cell toxicity or anoxic-

ischemic cell injury secondary to reduced uteroplacental blood flow. Gliosis of the brain stem in the respiratory centers has been a pathological finding in some cases of sudden infant death syndrome.[87]

Smokeless Tobacco

Another commonly used tobacco product with significant adverse medical effects is smokeless tobacco (both chewing tobacco and snuff). Estimates of the prevalence of ever using smokeless tobacco among high school students range as high as 30%. Approximately 20% of high school males have tried it within the last 30 days.[4] Smokeless tobacco use peaked in the late 1980s and has declined somewhat since then but remains relatively common.[4] Factors that influence the use of smokeless tobacco include gender (much more common in males than in females), region (rural more likely than urban), and race (Caucasians much more commonly than African-Americans or Hispanics). The pattern of use of smokeless tobacco is similar to that of cigarettes, with initiation of the habit beginning in the sixth through eighth grades and increasing thereafter.[88] About half of smokeless tobacco users also smoke cigarettes. Smokeless tobacco use is a readily available option for young adolescents who are experimenting with illicit drugs.[89]

The major health consequences of smokeless tobacco use in youth are related to the oral cavity and the cardiovascular system.[90-93] Periodontal disease is strongly associated with smokeless tobacco use. Caries and tooth abrasions may also be related. Cosmetic effects include halitosis and discoloration of teeth and fillings. All the effects of nicotine associated with cigarette smoking occur with smokeless tobacco use, including addiction, tachycardia, and acute increase in blood pressure. The major long-term health consequences of smokeless tobacco include increased risk for many oral cancers.

Lifestyle/Behavior

Onset of tobacco use among young people is primarily a social behavior.[94] Predictors of onset include sociodemographic, environmental, individual, and behavioral factors.[95] Modification of these factors is the basis for prevention and intervention. A young person who uses tobacco daily is likely to become addicted to nicotine.[96] Pharmacologic factors then become increasingly important, with cessation notably difficult in this age group.[97] Therefore, preventing onset and transition to regular smoking are the major aims of interventions with adolescents.

Tobacco use is most likely to be initiated in adolescence, a stage of life characterized by significant physiological, psychological, and social changes.[98,99] The social functions of tobacco use in our society, established in part by advertising, provide ways for adolescents to cope with these changes.[4] Being accepted by one's peers, asserting independence, feeling attractive, and signaling maturity are important to adolescents, especially so to those who have a low self-image, who are less academically successful, and who have fewer skills to cope with social pressures to smoke.[100,101] There may be some differences in why girls and boys begin to smoke. Girls who smoke tend to have good social skills whereas boys do not. Girls may also believe that smoking helps them control their weight.[102]

Tobacco use is associated with a range of health-compromising behaviors.[103] The 1988 Surgeon General's report concluded that smoking among adolescents is a risk factor for use of alcohol and illegal drugs.[104] The 1985 National Household Survey on Drug Abuse showed that 12- to 17-year-olds who smoked cigarettes in the previous 30 days were about three times more likely to have consumed alcohol, eight times more likely to have smoked marijuana, and 22 times more likely to have used cocaine in the past 30 days than adolescents who did not smoke cigarettes.[96] Cigarette smoking and use of smokeless tobacco appear to be entry-level or gateway drugs in a sequence of progressive drug use.[104] This does not imply that tobacco use *causes* illegal drug use; rather, those who used illegal drugs rarely did so without first smoking cigarettes. Similarly, cigarette smoking is associated with other rebellious, risk-taking, and deviant behaviors that together form a cluster of problem behaviors among adolescents.[101,105] Cigarette advertising reinforces these associations and has led to increased smoking by young women.[106] Smoking early in adolescence provides the foundation for participation in high-risk activities. Efforts to prevent tobacco use among adolescents might also prevent or delay the onset and development of subsequent problem behaviors associated with this syndrome.

International Aspects

Tobacco use should be recognized as a serious international pediatric health problem and a domestic health issue. The antismoking trend is seen in a number of countries, including Norway, Finland, Canada, New Zealand, and Australia. However, this is less true for Latin America, Asia, Eastern Europe, and Africa, where marketing has increased smoking rates and adverse health effects related to smoking have been observed or projected.[107-110] The fight against smoking in developing countries is hampered by a number of non–health-related political, economic, and educational factors.[107,111,112]

Interventions to Prevent Smoking

To young people, physicians are both medical experts and role models for appropriate health behaviors and therefore can be powerful communicators of nonsmoking messages.[113,114] However, these messages should differ according to the developmental stage of the child or adolescent. During infancy and early childhood the messages should be directed to the child's parents to not smoke. Parents should be informed that their smoking is a powerful influence on the subsequent smoking behavior of their child. Evidence of the effects of environmental tobacco smoke on pneumonia, bronchitis, asthma, and middle ear disease in children and sudden infant death syndrome should be given. Advice on cessation for parents who smoke should be made available. During childhood priority can be given to helping young people understand the health consequences of smoking, social influences to smoke, and ways to avoid smoking.[6] Among adolescents, promoting skills and intentions to remain nonsmokers and encouraging those who do smoke to quit become the paramount concerns. Advice

TABLE 1. Guidelines for Health Professionals to Prevent Onset of Smoking in Children[115]

Anticipate smoking risks associated with the child's developmental stage.
Ask about smoking by the patient or members of the patient's family.
Advise those who are trying, experimenting with, or smoking cigarettes to stop.
Assist in the smoking cessation process.
Arrange for follow-up on smoking status.

to youth should emphasize short-term effects on appearance, exercise performance, and physiology as well as long-term health benefits. The National Cancer Institute, the American Heart Association, and the American Academy of Pediatrics have developed guidelines for physicians to counsel parents on preventing smoking onset by children (Table 1).[115]

Discussion of smoking should be commonplace in the physician's office. Richards notes that "the words that a physician chooses to discuss smoking with a patient should be considered no less a therapeutic agent than the pharmacologic agent that the physician prescribes."[115] At present, advice to young people to not smoke is uncommon. Greater efforts to involve physicians, particularly those in pediatric and family medicine, are warranted.[116] Physician-based efforts for smoking cessation are effective. Therefore, the message for parents to quit should be integrated with the primary prevention message to children.[117-119]

Educational and communitywide programs to prevent or delay the onset of smoking among adolescents have shown promise over the last 15 years. It appears that complementary efforts at multiple levels in a community are needed for long-term effects. These efforts involve school-based prevention programs, youth-oriented mass media, parental and community programs, and policies to reduce availability, access, and acceptability of smoking in the community.[4]

Numerous studies of school-based smoking prevention programs have reported primarily positive findings for students who participated as young adolescents (sixth through eighth grades) in smoking prevention

TABLE 2. Eight Elements for a School-Based Smoking Prevention Program[125]

1. Classroom sessions should be held at least five times per year in each of 2 years in the sixth through eighth grades.
2. The program should emphasize social influences, short-term consequences, and refusal skills.
3. The program should be incorporated into the existing curriculum.
4. The program should be introduced during the transition from elementary to junior high or middle school.
5. Students should be involved in delivery of the program.
6. Parental involvement should be encouraged.
7. Teachers should be adequately trained.
8. The program should be culturally acceptable to each community.

TABLE 3. Framework for Public Policy Activities of the Coalition on Smoking OR Health

Advertising and promotion of tobacco products
Sale and distribution of tobacco products
Tax and pricing policy
Clean indoor air and environmental tobacco smoke
Regulation of tobacco products
Government tobacco-use prevention and cessation activities
Government support of tobacco

From the Coalition on Smoking OR Health *Framework for Public Policy Activities for 1993.*

programs based on social influences and skills-training modes.[120-124] Across these studies, reductions in weekly smoking ranged from 25% to 60% for 1 to 3 years following intervention. Programs based on social influences and skills-training models included discussions of the consequences of smoking, why young adolescents begin to smoke, social influences to smoke from peers and advertising, and ways to resist these influences. In 1987 the National Cancer Institute expert panel established the essential elements of effective smoking prevention programs (Table 2).[125] These essential elements have been largely supported by a recent meta-analysis.[124] Several additional research projects have demonstrated favorable long-term outcomes, at least to the end of the twelfth grade, when social influences and skills-training programs were augmented by booster sessions and communitywide quit-smoking programs[126] or by mass media involving television and radio spots designed to complement the school-based program.[127]

Community smoking policies that restrict access to cigarettes or the acceptability of smoking are an important component of the social environment that supports nonsmoking among young people.[128,129] They contribute to the perception by young people that nonsmoking is normal and public smoking is unacceptable. Most schools have policies on smoking; those with more restrictive policies for both students and staff have lower smoking rates.[130] National studies show public smoking restriction is associated with lower smoking rates.[129,131]

Adolescents report that obtaining cigarettes is easy, and these reports have been confirmed by studies of successful buying by underage teens.[132] There is preliminary evidence that a direct relationship also exists between tobacco access and smoking among young people.[133,134] Efforts to prevent access have included the regulation and banning of vending machines and greater enforcement and monitoring of age-of-sale laws, with preliminary data suggesting that these measures can reduce access to cigarettes and prevalence of smoking.[134,135] To date, however, no state in the United States has tobacco regulations that can be considered comprehensive.[136]

The Coalition on Smoking OR Health, a joint program of the American Heart Association, the American Lung Association, and the American Cancer Society, has targeted seven areas where government can be effective in limiting tobacco use and promoting health (Table 3).

Many initiatives may have an impact on smoking in youth. Current tobacco advertising campaigns are directed toward the young smoker, minimizing health effects and glamorizing tobacco use.[4,106] Programs that affect the nature of cigarette advertising such as restrictions on image advertising or complete bans could have a positive impact on smoking in youth. Paid cigarette advertising discouraging smoking and smokeless tobacco use could be effective in counterbalancing smoking industry efforts. Laws prohibiting the sale and distribution of tobacco products to those under the age of 18 can be more strictly enforced. A fee charged directly to the tobacco industry and linked to the number of cigarettes smoked by children and adolescents has been suggested as a disincentive to the industry.[137] Higher excise taxes will decrease tobacco use, particularly among the young who may have less discretionary money available for purchase of tobacco products. Regulations prohibiting smoking in public places, including schools and the workplace, will reduce passive smoke exposure, promote secondary prevention, and reinforce the message concerning the negative health effects of tobacco use. These negative health effects can also be enforced by more prominent labeling of tobacco products with regard to health hazards. Finally, the government can promote health education in the schools by helping develop and disseminate programs designed to discourage tobacco use.

Summary

This review defines the substantial pediatric morbidity from tobacco use, including health effects on the cardiovascular system, the respiratory system, the fetus and newborn, and risk-taking behaviors of adolescents.

More recent research suggests effects may extend to other areas, including reports that cigarette smoking decreases breast milk production in mothers, byproducts of tobacco use are transmitted in breast milk, exposure to passive smoking may alter children's intelligence and behavior, and passive smoke exposure in childhood may be a risk factor for developing lung cancer as an adult.[138-141]

Primary prevention is the most effective strategy to decrease the prevalence of smoking. Those who never smoke never become addicted to nicotine and never have to quit. Secondary prevention must also be emphasized, because children whose parents smoke are exposed to health risks and are themselves more likely to smoke in the future. Parental health can be improved by smoking cessation.[5] To accomplish the goals of primary and secondary prevention, the aggressive public health strategy directed at both parents and children should be expanded. This strategy requires the strong support of physicians, with emphasis on prevention in practice, support of public health initiatives, medical and public policy, and the conduct of high-quality research.

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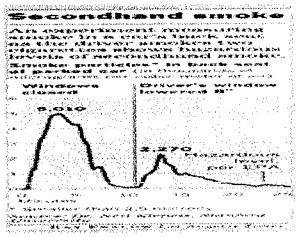
Putting smoking in cars to the test

California demonstration promotes the state's new ban on tobacco use in cars with minors.

By Mary Engel, Los Angeles Times Staff Writer
January 4, 2008

Smoking a cigarette in a car makes the air inside 10 to 30 times more toxic than the air outdoors on one of Southern California's most polluted days.

On Thursday, state officials put on a live demonstration of that health hazard to promote a new law that bans smoking in cars carrying minors.



Smoking experiment

Neil Klepeis, a Stanford University environmental health scientist, attached sensors to the dashboard of a 1999 Toyota Corolla parked on the lot of the Hollywood United Methodist Church. He attached additional sensors to a child safety seat in the back.

The sensors measured particulate pollution -- toxic, airborne pollutants found in cigarette smoke as well as in fumes from wood-burning stoves, diesel engines and other forms of combustion. The particles, about 30 times narrower than human hair, can lodge deep in the lungs and cause long-term health problems.

Such fine particles are particularly dangerous

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to children -- whose lungs are still developing -- and the elderly, said Dr. Mark Horton, director of the California Department of Public Health.

"Pound for pound, children breathe more air in than adults," he said. "Fine particulate matter is damaging to their lungs and can affect them forever."

Fine particles can cause or irritate asthma, bronchitis, pneumonia and ear infections, Horton said.

The demonstration showed how rapidly the particles build up in a small, enclosed space.

A volunteer smoker lit up in the driver's seat of the Corolla. Within 20 seconds, computer monitors showed particle levels in the front seat bounding beyond "unhealthy" and "very unhealthy" to "hazardous," the level that, if recorded outdoors, would draw a warning from the Environmental Protection Agency for residents to stay inside and not to exert themselves. Then, in less than a minute, the computer registered 30 times that hazardous level of particles.

The air in the back seat reached levels 10 times the "hazardous" level. When the smoker extinguished the cigarette and rolled down the window, the unhealthy levels lingered.

"You're creating a very large exposure for that child," said Klepeis, who last year published a study on secondhand smoke pollution in cars. "Smoke gets trapped in the back seat and can stay at high levels for a half-hour and at moderate levels for an hour or two."

In a repeat demonstration with the driver's side window open about 8 inches, the air still reached hazardous levels within a minute.

The new state law carries a fine of up to $100. Police officers cannot pull motorists over for smoking, but they can cite smokers if that offense is discovered in conjunction with another violation such as speeding.

"I'd be happy if we don't have to issue one citation," said state Sen. Jenny Oropeza (D-Long Beach), who sponsored the "Smoke-free Cars with Minors" law. "The objective of this new law is education. The objective is to get people to stop smoking in the car with kids."

California, which has been a leader in banning smoking in the workplace, restaurants and bars, follows Arkansas, Louisiana, Puerto Rico and Bangor, Maine, in banning smoking in cars with children. California's law is the most comprehensive because it covers passengers up to age 18, said Kimberly Belshe, secretary of the California Health and Human Services Agency.

In 2006 about 13% of California adults were smokers, down from 23% in 1988, in part because such laws have changed social norms, Belshe said.

Anthony Marquez, the volunteer smoker in the demonstration, approves of the new law and hopes it will prod him to quit smoking. He's tried quitting twice, he said.

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On latimes.com
To view a video of the demonstration, visit **latimes.com/smoking**.

CAMPAIGN for TOBACCO-FREE Kids

SECONDHAND SMOKE, KIDS AND CARS

"Exposure to secondhand smoke continues in restaurants, bars, casinos, gaming halls, ***vehicles***." – U.S. Surgeon General[1] [emphasis added]

"You can protect yourself and your loved ones by making your home and ***car*** smoke-free." – U.S. Surgeon General[2] [emphasis added]

State/commonwealth laws and key provisions:

Arkansas: No smoking with kids in a child safety seat (under 6 and under 60 pounds) in the vehicle. Enacted 4/10/06; Effective 8/3/06.
California: No smoking with a minor (under 18) in vehicle. Enacted 10/10/07; Effective 1/1/08.
Louisiana: No smoking with kids in a child safety seat (under 6 and under 60 pounds) in the vehicle. Enacted 7/5/06; Effective 8/15/06.
Maine: No smoking with kids under 16 in the vehicle. Enacted 4/10/08; Effective 10/1/08.
Puerto Rico: No smoking with kids under 13 in the vehicle. Enacted 3/2/06; Effective 3/2/07.

Local laws and key provisions:

Bangor, ME: No smoking with a minor (under 18) present in the vehicle. Enacted 1/8/07; Effective 1/19/07.
Keyport, NJ: No smoking with a minor present (under 17) in the vehicle. Enacted 4/24/07; Effective 2007.
Rockland County, NY: No smoking with a minor (under 18) present in the vehicle. Enacted 5/15/07; Effective 6/15/07.

Recent action by several states and territories to prohibit smoking in privately owned vehicles while children are present has brought significant attention to the issue of the risks to children associated with exposure to secondhand smoke, particularly in vehicles. While the research specific to children, secondhand smoke, and vehicles is limited, there is overwhelming evidence of the harms associated with exposure to secondhand smoke that is specific to children and specific to enclosed environments.

Harvard School of Public Health Study of Smoking in Cars with Kids[3]

A recent study by researchers at the Harvard School of Public Health found "alarming" levels of secondhand smoke were generated in just five minutes in vehicles under various driving, ventilation, and smoking conditions.

- The average levels of respirable particulate matter (the pollution inhaled from secondhand smoke) in the vehicles was actually higher than that found in similar studies of smoking in bars in several towns in eastern Massachusetts. In addition, the levels of particulate matter found in the vehicles exceeded those levels described by the U.S. Environmental Protection Agency as "unhealthy for sensitive groups" such as children and the elderly.
- The researchers found that the pollution levels detected "highlight the potentially serious threat to children's health presented by secondhand smoke in private cars under normal driving conditions."
- In addition to "alarming" increases of respirable particulate matter, the researchers also found a "significant increase" in levels of carbon monoxide. The researchers point out that carbon monoxide "is a poisonous gas, which may cause coma and death in large amounts, but among infants is known to induce lethargy and loss of alertness even in small quantities."

Based on their analysis, the researchers concluded that "smoking in cars under typical driver and traffic conditions provides potentially unsafe secondhand smoke exposure."

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Other Studies Warning of Secondhand Smoke Levels in Cars

- In 2006, a study published by *The New Zealand Medical Journal* found that smoking in a car with the window open produced air quality five times worse than even on the poorest air quality days in Auckland. Furthermore, it found that air quality was up to 100 times worse with all car windows closed. The study suggests adopting laws to make cars smoke-free in order to protect children and non-smokers from air pollution resulting from smoking in cars.[4]
- In 2006, researchers presented a study in which they found secondhand smoke in cars under all conditions tested reached unhealthy levels, even with ventilation. Extremely high levels of particulate matter were recorded in cars with tobacco smoke, putting all riders, particularly children, at an increased health risk.[5]
- A study published in *Ambulatory Pediatrics* measured the degree to which children with asthma living in urban areas are protected from secondhand smoke exposure. The results showed that among households with smokers, less than half (49 percent) maintained smoke free cars. The study concluded that protecting children from secondhand smoke exposure in their environment should be a public health priority.[6]

U.S. Surgeon General Statements on Children and Secondhand Smoke[7]

- "Secondhand smoke contains more than 250 chemicals known to be toxic or carcinogenic (cancer-causing), including formaldehyde, benzene, vinyl chloride, arsenic, ammonia, and hydrogen cyanide. Children who are exposed to secondhand smoke are inhaling many of the same cancer-causing substances and poisons as smokers."
- "Because their bodies are developing, infants and young children are especially vulnerable to the poisons in secondhand smoke."
- "Both babies whose mothers smoke while pregnant and babies who are exposed to secondhand smoke after birth are more likely to die from sudden infant death syndrome (SIDS) than babies who are not exposed to cigarette smoke."
- "Babies whose mothers smoke while pregnant or who are exposed to secondhand smoke after birth have weaker lungs than other babies, which increases the risk for many health problems."
- "Secondhand smoke exposure causes acute lower respiratory infections such as bronchitis and pneumonia in infants and young children."
- "Secondhand smoke exposure causes children who already have asthma to experience more frequent and severe attacks."
- "Secondhand smoke exposure causes respiratory symptoms, including cough, phlegm, wheeze, and breathlessness, among school-aged children."
- "Children exposed to secondhand smoke are at increased risk for ear infections and are more likely to need an operation to insert ear tubes for drainage."
- "The Surgeon General has concluded that the only way to fully protect yourself and your loved ones from the dangers of secondhand smoke is through 100% smoke-free environments."
- "If you are a smoker, the single best way to protect your family from secondhand smoke is to quit smoking. In the meantime, you can protect your family by making your home and vehicles smoke-free and only smoking outside."

American Academy of Pediatrics' on Children's Exposure to Tobacco Smoke[8]

The American Academy of Pediatrics has made the following conclusions regarding exposure of children to secondhand smoke:

- "Results of epidemiologic studies provide evidence that exposure of children to environmental tobacco smoke is associated with increased rates of lower respiratory illness and increased rates of middle ear effusion, asthma, and sudden infant death syndrome."
- "Exposure during childhood to environmental tobacco smoke may also be associated with development of cancer during adulthood."

The American Academy of Pediatrics recently adopted a resolution encouraging all its member state and local societies and chapters to:

"support and advocate for changes in existing state and local laws and policies that protect children from secondhand smoke exposure by prohibiting smoking in any vehicle while a legal minor (under 18 years of age) is in the vehicle."[9]

Campaign for Tobacco Free Kids, July 10, 2008

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[1] U.S. Department of Health and Human Services, *The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General*, U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 2006.

[2] The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General, U.S. Department of Health and Human Services, Children are Hurt by Secondhand Smoke, http://www.surgeongeneral.gov/library/secondhandsmoke/factsheets/factsheet2.html; The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General, U.S. Department of Health and Human Services, How to Protect Yourself and Your Loved Ones from Secondhand Smoke - http://www.surgeongeneral.gov/library/secondhandsmoke/factsheets/factsheet3.html.

[3] Rees VW, Connolly GN, "Measuring Air Quality to Protect Children from Secondhand Smoke in Cars," *American Journal of Preventive Medicine*, 2006 Nov; 31(5):363-8.

[4] Edwards R, et al., "Highly hazardous air quality associated with smoking in cars: New Zealand pilot study," *The New Zealand Medical Journal*, 2006 Oct, 119(1244), http://www.nzma.org.nz/journal/119-1244/2294/.

[5] Sendzik T, Fong G, Travers M, Hyland A, "The hazard of tobacco smoke pollution in cars: evidence from an air quality monitoring study," 13th World Conference on Tobacco or Health; 2006 July 12th–15th; Washington DC - http://www.arts.uwaterloo.ca/~gfong/smokefree/Sendzik-Car-WCTOH-2006.pdf.

[6] Halterman JS, et al., "Do Parents of Urban Children With Persistent Asthma Ban Smoking in Their Homes and Cars?" *Ambulatory Pediatrics*, 2006 Mar-Apr; 6(2):115-9.

[7] The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General, U.S. Department of Health and Human Services, Children are Hurt by Secondhand Smoke, http://www.surgeongeneral.gov/library/secondhandsmoke/factsheets/factsheet2.html; The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General, U.S. Department of Health and Human Services, How to Protect Yourself and Your Loved Ones from Secondhand Smoke - http://www.surgeongeneral.gov/library/secondhandsmoke/factsheets/factsheet3.html.

[8] American Academy of Pediatrics, Committee on Environmental Health, "Environmental Tobacco Smoke: A Hazard to Children", Pediatrics, Vol. 99, No. 4, April 1997.

[9] American Academy of Pediatrics, Resolution on Secondhand Smoke Exposure of Children in Vehicles (Resolution # LR2, (06) – 2006/2007 Annual Leadership Forum).